



Different magnetic resonance imaging features between MOG antibody- and AQP4 antibody-mediated disease: A Chinese cohort study

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ABSTRACT

Few studies have compared radiological features obtained on magnetic resonance imaging (MRI) between myelin oligodendrocyte glycoprotein antibody (MOG-ab)- and aquaporin 4 antibody (AQP4-ab)-positive patients. In this study, 77 MOG-ab and 92 AQP4-ab patients were enrolled. The results demonstrated that the brain MRI-based incidence of subcortical white matter lesions was higher in MOG-ab patients ($p < .000$) than in AQP4-ab patients and that the former therefore had a higher incidence of periventricular lesions ($p = .003$). The posterior limb of the internal capsule was more prone to lesions in MOG-ab patients ($p = .019$). Large lesions and U- or S-shaped lesions were also more frequent in MOG-ab ($p < .000$ and $p = .013$, respectively). Half of the MOG-ab patients had spinal cord involvement, and 36.5% presented with longitudinally extensive transverse myelitis (LETM). However, among the MOG-ab and AQP4-ab patients with spinal attack, there was no significant difference in the proportion with LETM ($p = .057$). In conclusion, a higher proportion of MOG-ab patients than AQP4-ab patients had brain lesions in white matter. Among MOG-ab patients who had an attack in the spinal cord, 65.5% also had LETM during the disease course. Conus medullaris lesions were rare in Chinese MOG-ab patients.

1. Introduction

Myelin oligodendrocyte glycoprotein (MOG) antibody (MOG-ab) and aquaporin 4 antibody (AQP4-ab) are two pathogenic auto-antibodies that can induce autoimmune demyelination in the central nervous system (CNS). MOG is a CNS-specific myelin protein that is mainly expressed in the outermost layer of myelin and has high immunogenicity. MOG-ab can induce oligodendrocyte injury and myelin damage, resulting in inflammatory demyelination in the CNS. MOG-ab-mediated disease has a broad clinical phenotype, with the majority of cases presenting with relapsing optic neuritis (ON), transverse myelitis (TM), neuromyelitis optica spectrum disorders (NMOSDs), and acute disseminated encephalomyelitis (ADEM) [1–3] but not classical multiple sclerosis (MS) [4,5]. AQP4-ab is known as the pathological landmark of NMOSD, and it can damage astrocytes and induce myelin damage and axonal loss [6]. However, while approximately 10–20% of NMOSD patients present as seronegative for AQP4-ab, 37% of AQP4-ab seronegative cases of NMOSD are MOG-ab-positive [4]. Recent studies have revealed that similar phenotypes also occur in other disorders,

such as ON, TM, and cerebral-stem encephalitis [7–9], making it difficult for neurologists to differentiate among these conditions.

Magnetic resonance imaging (MRI) is a useful instrument for distinguishing among CNS inflammatory demyelinating diseases, especially NMOSD and MS. MRI is often considered the first tool that should be used to distinguish demyelinating diseases caused by different auto-antibodies. In radiological imaging, AQP4-ab has a high specificity for lesions typical of NMOSD, such as longitudinal extensive transverse myelitis (LETM) and area postrema lesions [8]. MOG-ab tends to present as ADEM-like [1] and leukodystrophy-like phenotypes in pediatric patients and as ON in adult patients [10–12]. While the clinical characteristics associated with MOG-ab have been widely studied, the MRI characteristics of MOG-ab remain poorly defined.

In this study, we compared the abnormal lesions observed on cerebral, spinal cord, and optic nerve MRIs between MOG-ab-positive and AQP4-ab-positive patients with the aim of identifying MRI features that could potentially provide information that enables MOG-ab- and AQP4-ab-mediated diseases to be distinguished.

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Table 1
The clinical characteristics of MOG-ab patients and AQP4-ab patients.

	MOG-ab patients (n = 77)	AQP4-ab patients (n = 92)	P
Age at onset, mean \pm SD (range), y	26.8 \pm 16.2 (4–67)	30.3 \pm 14.7 (6–68)	0.073
Gender (M/F)	34/43	29/63	0.111
Follow-up, median (range), mo	32.4 \pm 19.3(8–72)	36.8 \pm 19.5(9–72)	0.131
Annual relapse rate	0.9	0.8	0.371
Serum IgG titer, median (range)	1:320 (1:50–1:1280)	1:100 (1:32–1:640)	0.002
Immunosuppressive Treatment	58/19	87/5	0.001

2. Materials and methods

2.1. Patients and antibody testing

In this retrospective study, we consecutively enrolled MOG-ab-positive patients and age- and sex-matched AQP4-ab-positive patients in the Neurology Department of the Third Affiliated Hospital of Sun Yat-sen University, China from January 2013 to January 2019. All patients had regular follow-up visits. The serum levels of MOG-ab and AQP4-ab were tested in each patient at least once per year during the follow-up period. Serum MOG-ab (immunoglobulin G to MOG) levels were tested using an in-house, cell-based assay in live cells transfected with full-length human MOG, as described in our previous study [10]. AQP4-ab (immunoglobulin G to AQP4) was detected using an indirect immunofluorescence assay (EUROIMMUN Medizinische Labordiagnostika AG, Lübeck, Germany) according to the manufacturer's instructions. Only patients seropositive for MOG-ab or AQP4-ab were included in this study. Patients with a disease phenotype considered by neurologists to be MOG-ab- or AQP4-ab-induced were enrolled.

This study protocol was approved by the Ethics Committee of the Third Affiliated Hospital of Sun Yat-sen University (Guangzhou, China). Written informed consent was obtained from all the participants.

2.2. MRI scanning

Brain, spinal, and optic nerve MRI scans were performed in all patients for routine clinical diagnostic purposes using a 3.0 T/1.5 T MR scanner (Discovery MR750/360, GE). Brain and optic nerve MRI data were obtained using a 3.0 T MR system. For brain MRI, axial T1WI (TR/TE/TI = 3500/24/943 msec), T2WI (TR/TE = 9500/93 msec), T2FLAIR (TR/TE/TI = 8400/145/2100 msec), sagittal T2WI, and contrast-enhanced axial, coronal, and sagittal T1WI (TR/TE = 1570/29 msec) data were analyzed. Axial and coronal T1WI (TR/TE = 430–470/16.9 msec), axial T2WI (TR/TE = 2500/86 msec), and T2FS (axial, sagittal, and coronal; TR/TE = 2500–4200/86–107 msec) data were obtained through the optic nerves without contrast, followed by sagittal, coronal, and axial T1FS (TR/TE = 423–670/9–17 msec). Sagittal T1WI (TR/TE = 500–600/13–16 msec), T2WI (TR/TE = 2000–3100/106–124 msec), STIR, and axial T2WI (TR/TE = 3800–5300/87–93 msec) data were obtained through the spine without contrast, followed by sagittal and axial T1WI obtained via postgadolinium administration (TR/TE = 170–300/4–6 msec) using a 1.5 T MR system.

MRIs obtained within two weeks of admission were included in this study [13]. All assessments were performed by two independent radiologists who were blinded to the patients' clinical features. Lesions were scored as large if their size exceeded 2 cm in any plane. The analyses of lesion locations and characteristics were the main topic of this research, and the MRI features as proposed by different diagnostic criteria for ADEM, MS and NMOSD were used in this study.

2.3. Statistical analysis

Statistical analyses were performed using the IBM SPSS, version 22.0 (SPSS, Chicago, IL, USA) software. The patients' ages are presented

as the mean \pm standard deviation with ranges. The length of LETM and immunoglobulin G titers are presented as medians with ranges. The clinical and radiological data were analyzed using the Mann-Whitney U test, Fisher's exact test, and Chi-squared test. A two-sided *p*-value $<$.05 was considered to indicate statistical significance.

3. Result

3.1. Demographics and clinical characteristics

The demographics and clinical characteristics of all patients are summarized in Table 1. A total of 77 MOG-ab patients and 92 age- and sex-matched AQP4-ab patients were enrolled in this study. Both groups were comprised predominantly of Chinese Han patients. There was no significant difference in the mean age at onset, the sex ratio, the median follow-up duration or the annual relapsing rate between the two groups. The median serum antibody titer at peak stage of relapse was 1:320 (1:25–1:1280) in MOG-ab patients and 1:100 (1:32–1:640) in the AQP4-ab group. At the last follow-up, 55 MOG-ab patients and 87 AQP4-ab patients had received immunosuppressive treatments (*p* = .001), including azathioprine, mycophenolate mofetil, rituximab and glucocorticoids.

3.2. Brain magnetic resonance imaging

In all, 169 brain MRIs were obtained in 77 MOG-ab patients, and 173 brain MRIs were obtained in 88 AQP4-ab patients during the acute stage and included in the analysis. Among the MOG-ab and AQP4-ab patients with normal cerebral imaging, 13 and 30, respectively, had completely normal MRIs throughout their available disease course (*p* $<$.001).

Table 2 summarizes the comparison of MRI features between MOG and AQP4-ab patients. On cerebral MRI, subcortical white matter lesions were found in the highest proportion of patients, including 49.4% (38/77) of MOG-ab patients and 12.5% (14/88) of AQP4-ab patients (*p* $<$.001). The incidence of periventricular lesions was also higher in MOG-ab patients than in AQP4-ab patients. Two MOG-ab patients had lesions that were perpendicular to the ventricular wall (Fig. 1). Juxtacortical white matter lesions were found in a higher proportion of MOG-ab patients (24.7%, 19/77) than AQP4-ab patients. The incidences of lesions in meningeal tissues, the internal capsule, the external capsule, the corpus callosum, the epithalamus, the insular lobe, the temporal lobe, and the cerebellar peduncle were all significantly higher in MOG-ab patients than in AQP4-ab patients. However, the number of lesions periventricular to the fourth ventricle (medulla and area postrema) was significantly lower in MOG-ab patients than in AQP4-ab patients. Interestingly, 15 of 18 MOG-ab patients who had internal capsule lesions also had lesions in the posterior limb of the internal capsule, and this incidence was higher than that found in the AQP4-ab group. There were no significant differences between the groups in the incidences of lesions adjacent to the aqueduct or third ventricle or in the cortex, dorsal thalamus, hypothalamus, hippocampus, midbrain, pons, or cerebellar hemisphere.

In addition, large lesions and U- or S-shaped lesions were significantly more numerous in MOG-ab than in AQP4-ab patients

Table 2
Comparison of MRI features between MOG-ab patients and AQP4-ab patients.

	MOG-ab patients	AQP4-ab patients	p
Brain MRI	n = 77	n = 88	
Abnormal	64	58	0.013
Meningeal	7	0	0.004
Juxtacortical	19	0	0.000
Subcortical	38	11	0.000
Periventricular to lateral ventricle	30	15	0.003
Internal capsule	18	7	0.008
External capsule	9	1	0.006
Adjacent to the fourth ventricle	4	31	0.000
Corpus callosum	14	4	0.006
Insular lobe	13	2	0.002
Midbrain	16	9	0.081
Pons	19	12	0.161
Medulla	13	37	0.001
Area postrema	2	30	0.000
Cerebellar peduncle	18	4	0.000
Spinal cord MRI	n = 52	n = 88	
Abnormal	29	78	0.000
LETM	19	66	0.000
Media length of LETM (range)	4 (3–12)	6 (3–19)	0.048
Conus medullaris	2	2	0.628
Centered	18	65	0.000
Eccentric	11	13	0.000
Enhancement	3	14	0.062
Swelling	10	39	0.003
Optic nerve	n = 42	n = 45	
Abnormal	28	32	0.817
Retrobulbar segment	19	8	0.010
Optic chiasma	3	1	0.331
Optic tract	2	2	1.000
Enhancement	22	24	0.770
Swelling	14	10	0.189

($p < .001$ and $p = .001$, respectively). ADEM-like lesions appeared in 14 MOG-ab patients (nine pediatric patients and five adult patients). Six MOG-ab patients presented with extensive and confluent hemispheric white matter lesions (Fig. 2). Two MOG patients had leukodystrophy-like lesions during their available disease course (Fig. 2). No classical Dawson's finger or T1 black hole was observed in this study. In addition, 10.9% (7/64) of the MOG-ab patients had cerebral lesion enhancement that was higher than that found among AQP4-ab patients ($p = .026$). Cloud-like enhancement appeared in 4 MOG-ab patients, and nodular enhancement appeared in 3 MOG-ab patients. Among the seven MOG-ab patients who had lesions in meningeal tissues, four showed meningeal enhancement, whereas no such occurrences were found in AQP4-ab patients. The frequency of cerebral atrophy was also higher in MOG-ab patients than in AQP4-ab patients ($p = .013$).

3.3. Spinal magnetic resonance imaging

Due to the retrospective nature of this study, we included only 52 MOG-ab patients who underwent 103 spinal MRIs and 88 AQP4-ab patients who underwent 191 spinal MRIs during the acute stage. Spinal cord lesions were observed in 55.8% (29/52) of the MOG-ab patients and 88.6% (78/88) of AQP4-ab patients. Among the MOG-ab patients, the locations of spinal cord lesions were as follows: 10 patients had isolated cervical lesions, four had isolated thoracic lesions, and 15 had both cervical and thoracic lesions. There were no significant differences between the groups in spinal cord lesion locations.

LETM was found in 36.5% (19/52) of the MOG-ab patients (Fig. 3) and 75.0% (66/88) of the AQP4-ab patients. However, among the patients with abnormal spinal cord MRIs, there was no significant difference in the LETM ratio ($p = .057$). The medial length of LETM was 4

segments in MOG-ab patients and 6 segments in AQP4-ab patients ($p = .048$). In addition, only 12.2% (5/41) of the MOG-ab patients but 67.1% of the AQP4-ab patients had LETM during their first episode ($p < .001$). In addition, up to 44.3% (39/88) of the AQP4-ab patients had spinal cord swelling during the acute stage, and this proportion was higher than that observed in MOG-ab patients (19.2%, 10/52).

No differences were found between the groups in spinal cord and pia mater spinalis enhancement. Central tube expansion was found in three MOG-ab patients and nine AQP4-ab patients, and this difference was not significant.

3.4. Optic nerve magnetic resonance imaging

Among the 42 MOG-ab patients with ON, 47 optic nerve MRIs were performed, and abnormal imaging was found in 66.7% (28/42) of these patients. Three MOG-ab patients had lesion involvement in only the left optic nerve, five showed involvement in only the right, and 20 were bilateral. Forty-five AQP4-ab patients with ON underwent 58 optic nerve MRIs, and 71.1% (32/45) of these patients showed abnormal lesions. Five AQP4-ab patients had lesion involvement only in the left optic nerve, five showed involvement only in the right, and 22 were bilateral.

No significant difference was found in the frequency of abnormal optic nerve MRIs. Approximately 67.7% (19/28) of the MOG-ab patients had lesion involvement in the retrobulbar segment of the optic nerves (Fig. 4), and this incidence was higher than that found among the AQP4-ab patients [14]. Interestingly, 52.4% (22/42) of the MOG-ab patients (Fig. 4) and 53.3% (24/45) of AQP4-ab patients showed optic nerve enhancement; while 21.4% (9/42) of MOG-ab patients showed perineural enhancement that extended into the soft tissues around the optic nerve, and this proportion was greater than that found among the AQP4-ab patients ($p = .050$).

In addition, 7.1% (3/42) of the MOG-ab patients showed involvement of the optic chiasm, and 2.4% (2/42) showed optic tract involvement; these incidences were not significantly different from those found in AQP4-ab patients. No significant difference in optic nerve swelling was observed between the two groups.

4. Discussion

MOG-ab and AQP4-ab are pathogenic antibodies in the CNS. Hence, ascertainment of a patient's serologic status can provide information valuable to the diagnosis and treatment of related demyelinating diseases. In this retrospective study, we summarized and compared the CNS MRI features of MOG-ab patients and AQP4-ab patients. The novel findings of this report include the following. First, a higher proportion of MOG-ab patients than AQP4-ab patients had brain lesions in white matter. Second, the posterior limb of the internal capsule was more prone to lesions in MOG-ab patients than in AQP4-ab patients. Third, half of the MOG-ab patients had spinal cord involvement and 36.5% of MOG-ab patients presented with LETM during their disease course. Fourth, conus medullaris lesions were rare in Chinese MOG-ab patients.

Among the brain MRIs evaluated in this study, 83.1% of those obtained in MOG-ab patients showed brain lesions and locations that were different from those found in AQP4-ab patients, especially with regard for subcortical and juxtacortical white matter lesions [15–17]. Several previous studies had reported on the predominance of subcortical, nonenhancing lesions and juxtacortical lesions in pediatric MOG-ab patients with ON [15] or seizure [3,16]. Although radiological manifestations are observed in a high proportion of these patients, these lesions are still thought to be atypical of MOG-mediated disease. In contrast, studies have verified that the inflammatory cortical brain lesions found on MRI are responsible for the seizures observed in MOG-ab patients [18] but not those observed in AQP4-ab patients [17]. Our previous study revealed that most MOG-ab patients have cortical/subcortical brain lesions and that these lesions are associated with seizures

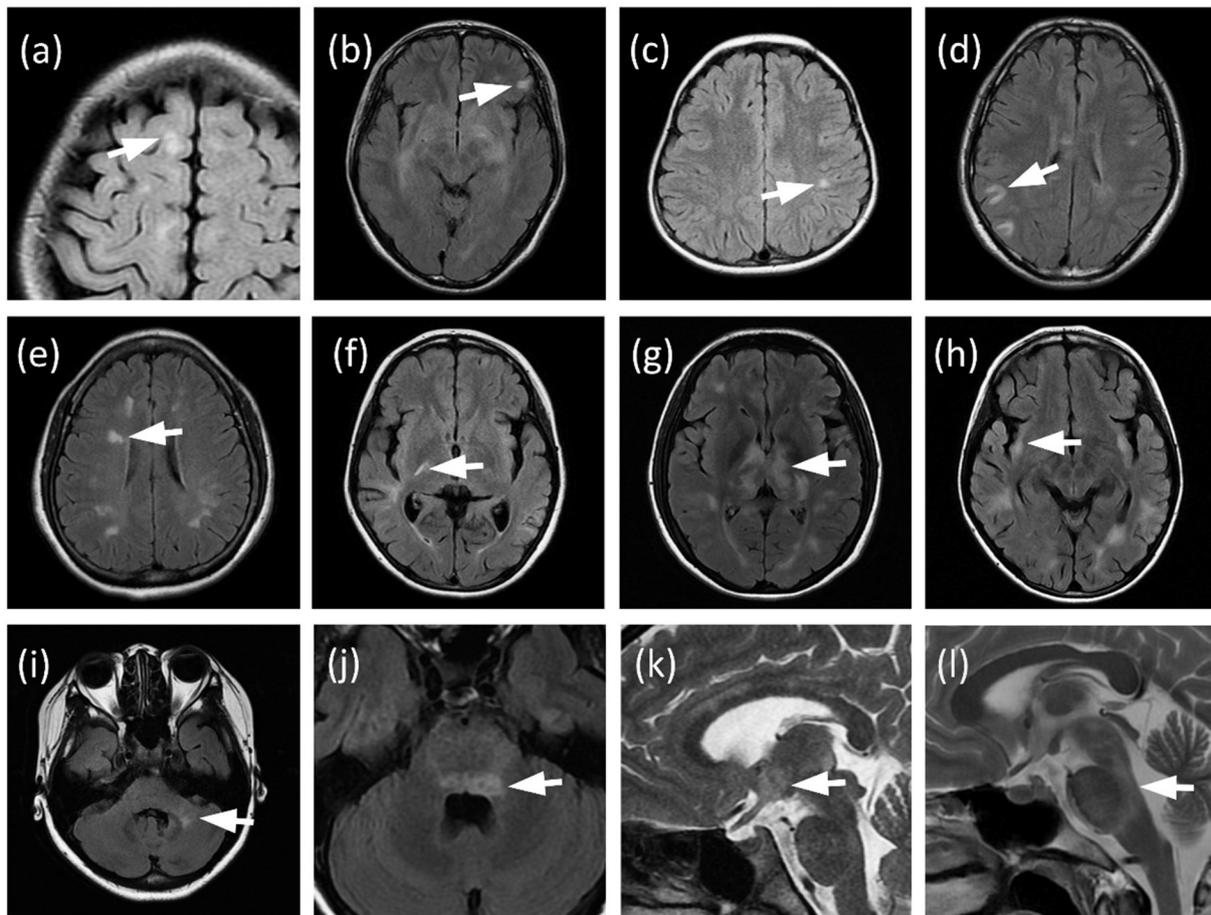


Fig. 1. Representative brain lesions in MOG-ab patients in axial T2 weighted (a-j) or sagittal FLAIR (k, l) magnetic resonance imaging. (a) Cortical lesion in right frontal lobe. (b) Juxtacortical white matter lesion. (c) Subcortical white matter lesion. (d) U-shaped fiber lesion. (e) Lesion was perpendicular to the ventricular wall. (f) Lesion in the posterior limb of internal capsule. (g) Lesion in thalamus. (h) Lesion in insular lobe. (i) Lesion in cerebellar peduncle. (j) Lesion adjacent to the fourth ventricle. (k) Hypothalamus lesion. (l) Lesions in midbrain and pons. Arrows pointed to the locations of brain lesions.

and encephalopathy [19]. Lesions in the hypothalamus were previously proposed to be specific to AQP4-ab pediatric patients, but we found that four MOG-ab patients (one child and three adults) also had hypothalamic involvement. The brain lesions observed in AQP4-ab patients were highly associated with areas with high AQP4 expression, especially periaqueductal and third and fourth ventricular lesions. In this study, we found that the frequency of fourth ventricular involvement was higher in AQP4-ab patients (34.1%) than MOG-ab patients, in accordance with a previous study [20], whereas lesions in the area postrema were only occasionally observed (in two MOG-ab patients).

Notably, 23.4% of MOG-ab patients showed lesions in the internal capsule, and 83.3% of these involved the posterior limb of the internal capsule. Lesions in the posterior limb of the internal capsule are thought to be a useful feature in distinguishing MS from NMOSD as they were found in 23.5% of NMOSD patients in a Korean study [20]. In our study, only 6.8% of the AQP4-ab patients had lesions in the posterior limb of the internal capsule, and this rate was significantly lower than the 19.5% found in MOG-ab patients. The difference between these results could be explained by differences in inclusion criteria and sample sizes. In addition to the lesions observed in the internal capsule, 11.7% of MOG-ab patients also had lesions in the external capsule, a finding not frequently reported in the literature.

Periventricular lesions and corpus callosum lesions are frequently observed in MS and AQP4-ab-positive NMOSD [20]. In our study, these lesions and lesions in the deep gray-matter lesions [21] were more frequent in MOG-ab than in AQP4-ab patients. On brain MRI, 39.0% of MOG-ab patients showed lesions in the periventricular areas of the

lateral ventricle, and this proportion was higher than that observed among AQP4-ab patients. In addition, two MOG-ab patients had lesions perpendicular to the ventricular wall that were absorbed after clinical treatment. In clinical practice, MS may be diagnosed in a certain segment when juxtacortical lesions and periventricular lesions appear and show dissemination over time and space in MOG-ab patients [9,15]. Even though some patients showed MS-like characteristics on MRI, no patient had been diagnosed with MS at the end of follow-up [22].

Large lesions in brain MRIs are common in MOG-ab-mediated disease. Recent data suggests that MOG-ab could be detected in a subgroup of pediatric childhood demyelinating disorders, presenting with ADEM-like lesions [23]. In our study, ADEM-like lesions appeared in 14 MOG-ab patients, and 64.3% were pediatric patients. In addition, 7.8% of MOG-ab patients presented with extensive and confluent hemispheric white matter lesions. All the lesions were tumefactive and resulted in clinical manifestations indicating intracranial hypertension in several patients. Leukodystrophy-like lesions, which have previously been described in pediatric patients, were also observed. This phenotype is rare and profound in children and might hint that the myelinating brain is susceptible to MOG-ab-mediated disease [12]. Brain lesion enhancement was found in 9.1% of the MOG-ab patients. Cloud-like enhancement and nodular enhancement were the main patterns observed in MOG-ab patients, as previously reported in NMOSD [20]. Focal brain atrophy appeared in two MOG-ab patients and was localized in the lobe where previous extensive hemispheric white matter lesions had appeared. MOG-ab patients with large lesions, such as those that were ADEM-like and leukodystrophy-like, can potentially develop

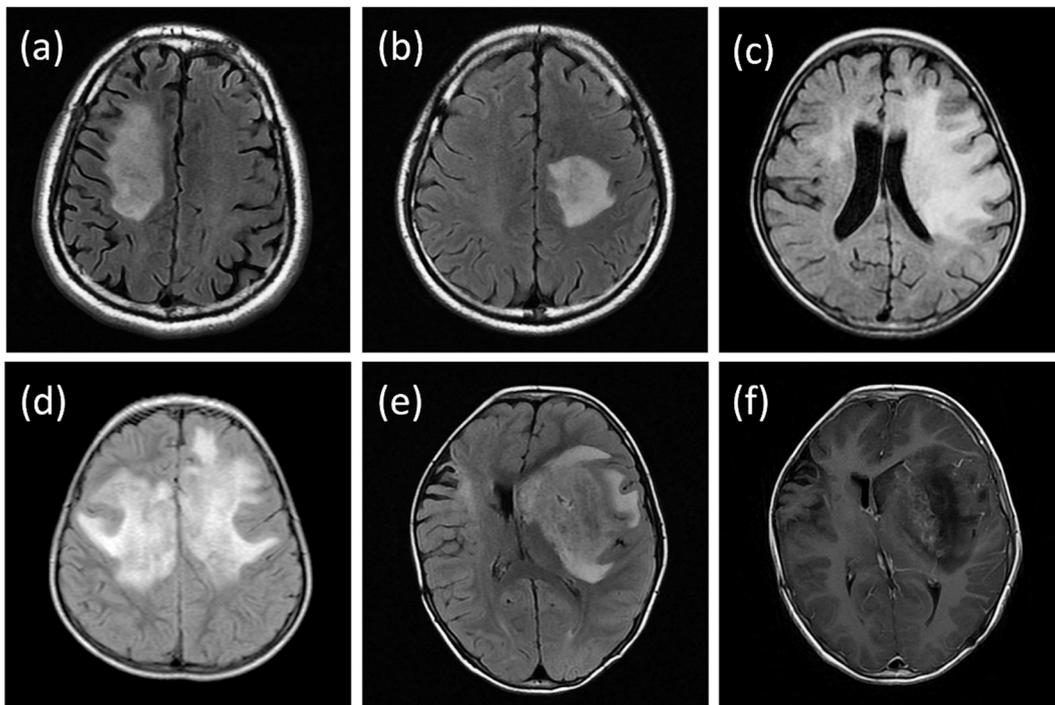


Fig. 2. Examples of large lesions and 'leukodystrophy-like' lesions in MOG-ab patients in axial FLAIR MRI. (a, b, e) Large lesions with diameter > 2 cm in brain MRI. (c, d) 'leukodystrophy-like' lesions in brain MRI. (f) Large lesion with gadolinium enhancement in a child patient. Images of (e) and (f) were from the same patient.

neuroaxonal injury [12]. According to a recent study, half of MOG pediatric patients with an abnormal brain MRI progressed with cognitive problems [12].

In addition to abnormal brain MRI results, myelitis also frequently occurs as the initial manifestation in MOG-ab-mediated disease [24]. Myelitis can present with ON [21] and brain involvement that manifests as NMOSD and ADEM-like phenotypes [7]. In our study, 55.8% of the MOG-ab patients had spinal lesions throughout the disease course, and this proportion was lower than that observed in AQP4-ab patients. Among MOG-ab patients with spinal lesions, 65.5% presented LETM, and this proportion was not significantly different from that observed in AQP4-ab patients, consistent with another study (79%) [7]. This result might indicate that MOG-ab patients have the same high risk of LETM progression as has been reported in AQP4-ab patients. However, MOG-

ab patients were more likely than AQP4-ab patients to have two non-contiguous lesions [9]. The appearance of oligodendrocytes expressing MOG has been verified in gray matter in both the spinal cord and the brain [25]. Lesions restricted to the spinal cord gray matter appeared in 32.0% of MOG-ab patients, a result close to the 29% reported in previous MOG-TM studies [7,26]. However, only 3 MOG-ab patients in this study showed spinal cord enhancement that was restricted to the gray matter. A recent study demonstrated that compared with AQP4-ab and MS myelitis patients, in MOG-ab patients, lesions restricted to the gray matter that showed a lack of enhancement were a discriminating feature [7]. While most of the MOG-ab patients exhibited prominent motor deficits, and 30% of these patients were wheelchair-dependent at nadir, more MOG-ab patients than AQP4-ab patients achieved a good recovery and good long-term outcomes [7].

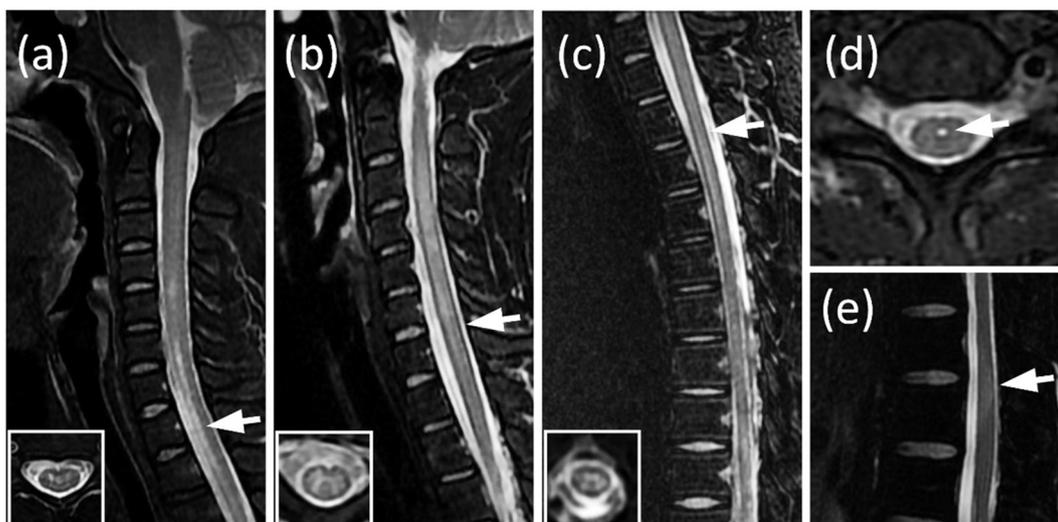


Fig. 3. Representative spinal cord lesions in MOG-ab patients in T2WI magnetic resonance imaging. (a) Long segment myelitis in spinal cord. (b, c) LETM in the cervical cord and thoracic cord. (d) Expansion of central tube showed on axial imaging. (e) Lesion in the conus medullaris. Arrows pointed to the locations of lesions.

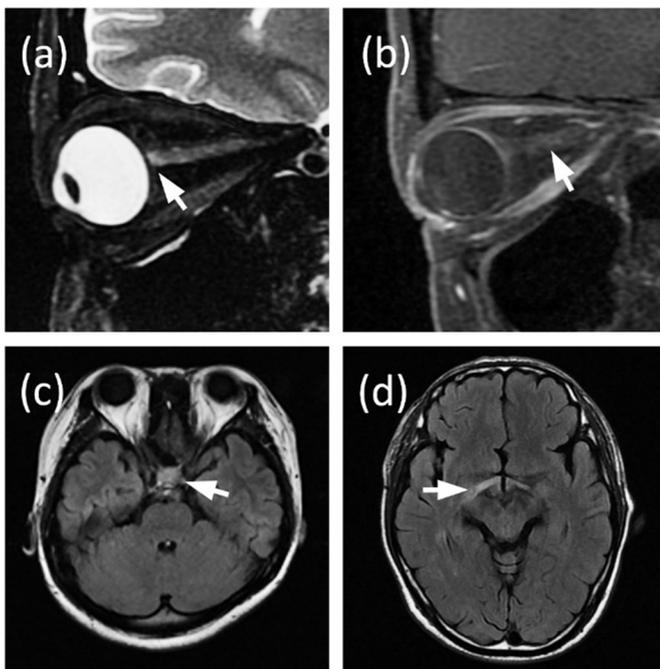


Fig. 4. Examples of lesions found in MOG patients in magnetic resonance imaging. (a) Lesion found in the retrobulbar segment of optic nerves in T2 fat suppression. (b) Enhancement in optic nerve after gadolinium administration. (c, d) Lesions in optic chiasma and optic tract on FLAIR imaging.

ON was the main clinical phenotype observed in MOG and AQP4-ab patients with frequent recurrent episodes. > 80% of adult MOG-ab patients had ON during their disease course, similar to the results obtained in our study. MOG-ab patients tended to have more frequent attacks but with better visual recovery [22,27]. Only 66.7% of MOG-ab patients and 71.1% of AQP4-ab patients with ON showed abnormal results on optic nerve MRI, although all of these patients were in the acute stage. In addition, 71.4% of MOG-ab patients and 68.8% of AQP4-ab patients who had abnormal MRI results showed bilateral involvement in the optic nerve. This result is similar to those reported in other studies [14,28]. Longitudinally extensive optic nerve lesions were mainly seen in AQP4-ab patients, but lesions in the retrobulbar segment of the optic nerves were more often observed in MOG-ab than in AQP4-ab patients [14].

There are several limitations to this study. First, this study was a single-center, retrospective study. Patients were enrolled from a single university hospital in China and were drawn from a single ethnic population. The results therefore need to be verified in prospective, larger-scale, multicenter studies. Second, this study was designed to describe and compare the MRI features of MOG-ab patients with those of age- and sex-matched AQP4-ab patients. We acknowledge that there may have been a risk of selection bias. However, the results of our study still represent an important contribution to recognizing the different features of these diseases.

5. Conclusion

MOG-ab-mediated disease exhibits different radiological features than are observed in AQP4-ab-mediated disease. MOG-ab patients had wider involvement in the CNS, especially the brain. Subcortical and juxtacortical white matter lesions and lesions in the posterior limb of the internal capsule were more frequently observed in MOG-ab patients than in AQP4-ab patients. In addition, MOG-ab patients sometimes developed LETM, similar to AQP4-ab patients. However, conus medullaris lesions were rare in Chinese MOG-ab patients.

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